infection with inflammation. It has communities of microorganisms, which raises interest in terms of inflammation reaction and the microbial burden in fields like cardiology and obstetrics. This means we all have a common interest in the aetiological factors.

What information does current research give us about the microbiological interactions in biofilm?

One first has to understand that this is not simply about a small group of bacteria, but perhaps about the total microbial burden and the immune reaction to this burden. For example, Streptococci are early colonizers who may play a role in other diseases. In the field of periodontology, we haven’t paid much attention to this thus far.

Do you see a need for improved cooperation between dentistry and medical science?

I already work with cardiologists and gynecologists at the University of Seattle and with physicians from Sweden and Bern in Switzerland because as a dentist, I have microbiological information they may not have. You see, there is close cooperation in the fields of periodontology, immunology and social behavior.

Despite this cooperation, we may be too late in some cases. Periodontal treatment of a 70-year-old patient will yield no improvement, but we might be able to treat 50-year-old patients with the help of a special diet and improved oral hygiene, or with antimicrobial and anti-inflammatory treatment methods that influence cardiovascular conditions.

I also see a lot of potential in cross-sectional intervention studies. In these studies we observe healthy and sick patients and examine their dental conditions and the way in which these conditions and other medical conditions change because of treatment.

Can you give an example?

One could look at epidemiological studies of Jönköping County (a province in Sweden), conducted from the 1970s until today. In 1970, almost 80 percent of the county’s residents had some form of periodontitis, and a rather small number, about 15 percent, suffered from severe periodontal disease. The first group of people do not have periodontitis nowadays, which indicates a significant change over the last 50 years. However, the group with severe periodontitis has not changed.

Why is that?

In my opinion, because of the Swedish health care system. Patients with periodontal disease underwent treatment, but in the group with severe periodontitis, these methods were not successful. Current methods in periodontology are not sufficient in my opinion. Mechanical treatments, such as scaling and root planing, are not able to remove bacteria in patients that already have symptoms of disease. It could be that these treatments do the opposite, and cause coronary embolism. In addition, there is immune reaction.

There are two studies, conducted in Australia and the United Kingdom, that observed blood circulation in the arms and found that the level of a certain protein increased shortly after periodontal treatment (between 2 mg/l and 15–20 mg/l). The levels decreased after a while, but they did not return to normal. Therefore, the separation did not result in elimination of inflammation factors and thus wasn’t successful in my opinion. One cannot expect to treat patients with risk of cardiovascular disease or preterm birth successfully because the studies show that the risks basically remain the same.

We generally need more knowledge of the relationship between and the role of microorganisms and immune defense systems. In addition, there are socioeconomic and genetic factors that we cannot influence at all. My hope is that politicians put more effort into supporting joint academic research between dentistry and medical science.

Is there a lack of support for such research?

I think there are enough funds available for medical research, but it is very difficult for dentistry to compete with medical science in that respect. The reason is that it is a smaller discipline. But improved cooperation between medical science and dentistry could reduce the risk of preterm births and cardiovascular diseases.

What role can the industry play?

Implants are very interesting as a replacement for natural teeth, but we do not know much about the mechanisms between peri-implantitis and systematic diseases. We do know that Staphylococcus aureus, for example, sticks on titanium inside biofilm, and causes inflammation, which was proved in medical studies involving titanium prosthetics in hand and joint replacement.

In my opinion, because natural teeth and implants are not the same, conditions for the colonisation of bacteria on implant surfaces are different from those of teeth. It is also much more difficult to clean an implant. The problem is that the industry propagates very high success rates of their products, which is too short-sighted in my opinion.

In Sweden, for example, all joint implants have to be approved. We therefore, one knows exactly how many have been implanted, and how many of those were successful. In dentistry, such a list does not exist, and we therefore do not know how many implants have been successful thus far.

In addition, it must be noted that it takes 20 years for natural teeth to develop periodontitis. The first implants are about 20 to 50 years old, and only now can one see how they have developed. At first, only patients without risk of peri-implantitis received implants; yet, all dentists worldwide can place implants, even if he or she is not qualified. There are certainly ulcerative motives involved here, and it is apparent that some patients have received implants at all. There is much potential for mechanical and technical failure. But how does one separate successfully treated patients from patients that suffer from infections, inflammation and pathogenic organisms? In this case, analysis was not very accurate.

This issue will continue to be of concern to dentistry. What can be done?

In Bern, we have been using the same implant system for years. About 1,000 implants were placed in the last 10 years, so we have a follow-up time ranging from five to seven years. After such a period, one can conclude how successful treatment was through microbial, ethnological, clinical or socioeconomic studies that determine the success of a treatment.

Multiple center studies could also help to identify the different mechanisms, and help us choose patients with minimal risk or no risk at all of implant failure. Then we might be able to find methods to treat peri-implantitis. I believe we also have to consider antibiotics and anti-inflammatory compounds. Cleaning implants with hand instruments and toothbrushes at home isn’t enough.

Another interesting aspect is the relation between tooth implants and periodontal inflammation or the so-called peri-implantitis.

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What role can the industry play?

This is a very interesting question. I recently discussed this with a colleague from Stockholm, and we both agreed that fluoride toothpaste, developed mainly by the industry and not by universities, was the biggest breakthrough in the 1980s and 1990s. This example shows that research conducted by the industry can be very successful.

Personally, I see no problem in this because implant companies make a lot of money and should be responsible for pouring some of their research money into research institutes, instead of constantly developing new implant systems. This could lead to a better understanding of the mechanism of many successful implants and implants that fail. It will be up to governments and health authorities to introduce control mechanisms for these processes.